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Coronary Vasospasm Mimicking Acute Myocardial Infarction

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Authors' contributions

This work was carried out in collaboration between all authors. Author WJ designed and wrote the first draft of the manuscript. Author SPJ revised the case report. Author Shilpi Singh managed the literature searches. Authors Shuvendu Sen and AY has reviewed and finalized the case report. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Chest pain frightens many people into thinking that they might be having a heart attack. There are many causes of chest pain, few of which are life-threatening. One of the causes of chest pain is vasospasms, which is transient in nature and prolonged episodes can lead to tissue necrosis. Therefore, a prompt diagnosis and treatment is vital to the management. We are reporting a case of a young female presented with chest pain with no history of drug use, previous episodes, palpitations, and hypertension being her only risk factor found to have elevated Troponin I and was diagnosed with non-ST elevation myocardial infarction (NSTEMI), later found to have coronary vasospasm.

Keywords: Coronary vasospasm; NSTEMI; troponin; myocardial infarction.

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1. INTRODUCTION

Since its introduction in the early nineties, troponin assays have been implemented in most emergency facilities as point of-care tests, or are offered on emergent basis by the hospital laboratories [1]. Troponin still is a remarkable marker in patients presenting with myocardial cell injury. Troponin elevation in the absence of an Acute Coronary Syndrome has significant prognostic value, both short-term and long-term, given a resourceful tool in the treatment of Myocardial Infarction (MI) [2]. Acute myocardial infarction is defined as myocardial cell death due to prolonged myocardial ischemia [3]. Clinical classifications of different types of myocardial infarction are spontaneous MI related to ischemia which can be due to a primary coronary event such as plaque, erosion, rupture, fissuring, dissection or secondary to ischemia due to increased oxygen demand or decreased supply. Coronary vasospasm is an important etiology of ischemic heart disease. Previous reports have proven that severe attacks of vasospasms may progress to myocardial infarction or sudden death, although this is rare [4]. Treatment should be established promptly. ST-T changes in an ECG can be due to spasms as well. Coronary vasospasm is a diagnosis based on risk factors seen in patients who are smoker or ex-smoker, have a history of hyperlipidemia and hypertension in spite of elevated troponin. Some risk factors for hypertension in our case that were not present were smoking, African American race, diabetes, obesity, and alcohol intake. In any case of vasospasm, one should explore the possibility of ischemia, acute coronary syndrome, arrhythmia, and sudden cardiac arrest. In our scenario, we present a young female patient complained of constant chest pain with no alleviating or aggravating factors, no diaphoresis, nausea, shortness of breath, palpitations, and no previous episodes of chest pain. She had hypertension as the only significant risk factor for Myocardial Infarction. A diagnosis in our patient was made based on symptoms, ECG showed ST-T changes, and high troponin levels. Spasm of the right coronary artery resolved with nitroglycerin during cardiac catheterization. Therefore, a patient who experiences a prolonged vasospasm who is not promptly diagnosed and treated can have ischemic cell death.

2. CASE PRESENTATION

A 37 year old Hispanic female with significant history for hypertension presented to the emergency room with two days history of intermittent chest pain. However, on the day she presented to emergency department it was constant, 9/10, retrosternal, with radiation to the left arm; without any relieving or aggravating factor. It was associated with diaphoresis and nausea. Patient denied palpitations, shortness of breath, or sore throat. Patient was taking Lisinopril for hypertension. Patient was born in Dominican Republic and had family history of diabetes mellitus. Patient denied any smoking, alcohol, drug use, recent travel, and any foreign body or sick contacts. On physical examination, blood pressure was 136/90 mm Hg, pulse 80/min, temperature of 98ºF, and respiratory rate 16/min with SPO_2 of 99%. Remainder of physical exam was unremarkable without any significant findings. Laboratory data showed Hemoglobin 14 g/dL, WBC 8.3 K/uL, platelets 290 K/uL, INR 1, PTT 26 sec, D-dimer is less than 0.3, Glucose 116mg/dL, BUN 11mg/dL, creatinine 0.6mg/dL, calcium 9.8mg/dL, albumin 4.6g/dL, total protein 7.7g/dL. Sodium 135mmol/L, potassium 3.5mmol/L, chloride 97mmol/L, bicarbonate 28mmol/L. Anion GAP10mmol/L. Alkaline phosphatase 60U/L, total bilirubin 0.3mg/dL, AST 22U/L, ALT 19U/L. CK is 218IU/L, CKMB 11.52IU/L. Troponin I is 1.1ng/mL (Normal <0.30ng/mL). Patient's ECG showed nonspecific ST-T changes (Fig. 1). Patient was started on heparin infusion consider that the patient is having acute myocardial infarction. Patient's potassium was replaced and corrected potassium level was 3.7mmol/L but the repeat ECG showed same ST-T changes and patient's second set of troponin I was reported 5.65ng/mL. Despite all patient remained in chest pain. Patient was taken for cardiac catheterization. Coronary angiography was performed which showed right coronary artery (RCA) spasm which resolved after intra coronary injection of nitroglycerine (Figs. 2 and 3). Patient's Echocardiogram was normal. She was discharged home on amlodipine and aspirin once the troponin started trending down. Upon follow up, patient remained asymptomatic.

3. DISCUSSION

Compared with patients without coronary vasospasm, patients with coronary vasospasm had a higher frequency of elevated cTnI and transient ECG ST-T changes, ST elevation or depression (23% vs 2%, *P* _ .001) and a lower left ventricular ejection fraction [4].

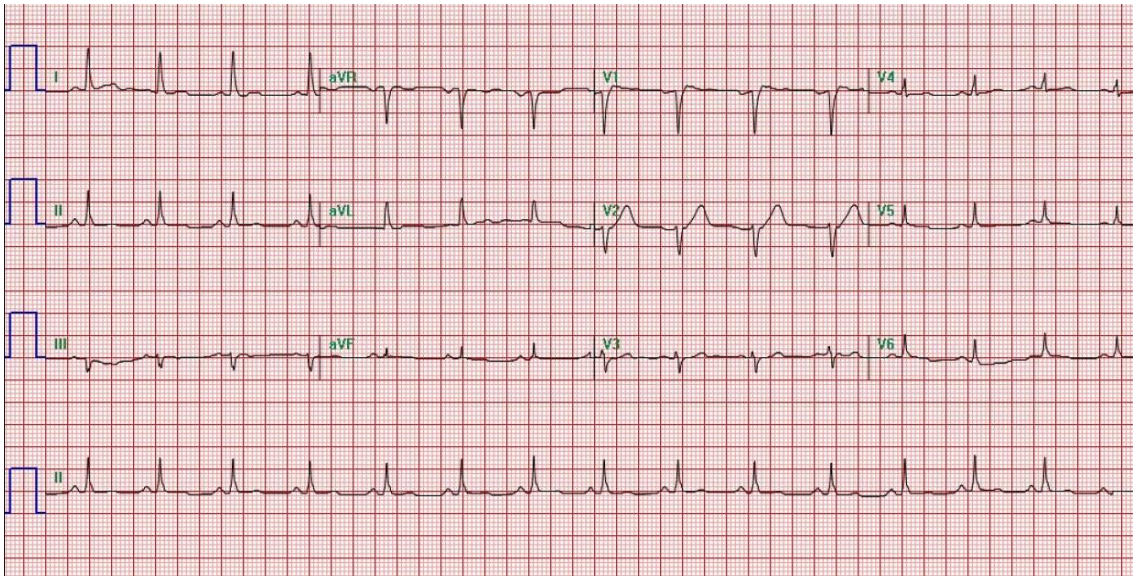

Fig. 1. Showing nonspecific ST-T changes

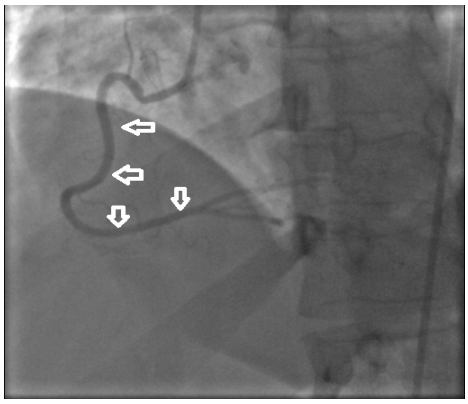

Fig. 2. Showing vasospasm of the right coronary artery

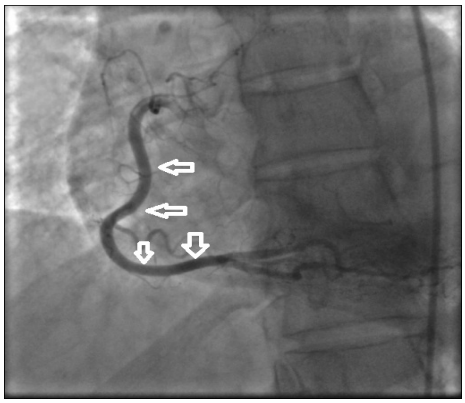

Fig. 3. Showing post nitroglycerine injection that spasm has resolved

Coronary vasospasm is commonly associated with a fixed atherosclerotic lesion as mentioned with risk factors but can occasionally present in normal arteries [4]. This case report emphasis that vasospasm can also give rise to elevated cardiac troponin I levels and ST-T changes in normal coronary arteries. Etiology of vasospasm is not fully understood but it can be related to risk factors such as hypertension, hyperlipidemia and smoking. Nitroglycerine and effective time management was the key factor to the resolution of symptoms in our case. Calcium channel blockers are the choice of treatment once the acute phase has resolved. Even though troponin level is a remarkable marker, it should not always be used as the only source of diagnosing myocardial infarction because many different other causes can lead to raised levels. Some of the causes which can give elevated troponin I are pulmonary embolism, myocarditis, and trauma from Cardiopulmonary resuscitation, seizures, end stage kidney failure, and increased intracranial pressure. Therefore before considering vasospasm, an approach should be used to rule out all possible causes of coronary artery disease (CAD). A scenario to consider vasospasm will be resting chest pain with ECG changes, and troponin elevation. Most patients are usually of young age with no significant risk factors and normal coronaries on cardiac catheterization. But there are also some racial differences exist between Oriental and Western countries in coronary vasospasm [5]. Onset of pain is usually at rest and sometimes at night

and not associated with exertion. Stress test will be negative in these patients. ECG changes are only expected when patients are experiencing chest pain. Stimulating tests with ergonovine can be useful during cardiac catheterization to induce vasospasm which can aid in the diagnosis.

4. CONCLUSION

Prinzmetal angina or coronary artery vasospasm can present with or without ST elevation in the electrocardiogram mimicking acute MI. Thus, not only cardiologists but primary care physicians should also be aware of the fact that coronary vasospasm can cause chest pain with or without elevation of troponin.

CONSENT

All authors declare that 'written informed consent was obtained from the patient (or other approved parties) for publication of this case report and accompanying images.

ETHICAL APPROVAL

Not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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